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ABSTRACT

A survey of the evidence shows that some degrees of malnutrition is relatively widespread among poor children. However, the effects of inadequate nutrition on growth and mental development depend to a large extent on the severity, the timing (pre and postnatal), and the duration of the nutritional deprivation. The data are inadequate on the true prevalence of malnutrition among children in this country, but there is even less information about its onset or about its severity and quality. The absence of such knowledge reflects not the absence of the problem, but the lack of attention devoted to it. There is strong indication that nutritional factors at a number of different levels contribute significantly to depressed intellectual level and learning failure. Moreover, an adequate state of nutrition is necessary for good attention and for appropriate and sensitive responsiveness to the environment. Further, women who were malnourished as children are more likely to have disturbed pregnancies and children of low birth weight and increased risk of neurointegrative abnormality. It must be recognized, however, that improvement of nutrition alone cannot fully solve the problem of intellectual deficit and school failure. Rather, an overall effort to improve the condition of disadvantaged children is required. (Author/NH)

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DESIGNS AND PROPOSAL FOR EARLY CHILDHOOD RESEARCH: A NEW LOOK: MALNUTRITION, LEARNING AND INTELLIGENCE

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Malnutrition, Learning and Intelligence

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INTRODUCTION:

Research on the relation of nutritional factors to intelligence and learning has burgeoned over the past decade. Its resurgence after a period of nearly thirty years of quiescence which followed Patterson's (1930) review of studies conducted in the first three decades of the century reflects a number of social and historical currents. Newly emerging nations as well as aspiring underprivileged segments of the population in more developed parts of the world have increasingly come to be concerned by the association of social, cultural and economic disadvantage with depressed levels of intellect and elevated rates of school failure. Attention has variously been directed at different components of the combined syndromes of disadvantage and poverty in an effort to define the causes for such an association. Sociologists, psychologists and educators have advanced reasons for intellectual backwardness and school failure relevant to their particular concerns. They have pointed to particular patterns of child care, cultural atmosphere, styles of play, depressed motivation, particular value systems, and deficient educational settings and instruction as factors which contribute to lowered intellectual level and poor academic performance in disadvantaged children. The importance of such variables cannot be disputed and studies and findings relevant to them expand our understanding of some of the ways in which poor achievement levels are induced. However, it would be most unfortunate if, by recognizing the importance of these situational, psychological and experiential components of the syndrome of disadvantage, we were to conclude that they represented the whole of the picture or even its most decisive components. Any analysis of the content of poverty and disadvantage rapidly brings to our notice the fact that these negative features of the behavioral and educational environment take place within the pervasive context of low income, poor

housing, poor health and, in general, defective circumstances for the development of the individual as a biologic organism who interacts with the social, cultural and educational circumstances.

Such considerations inevitably cause us to expand the range of our concern to include a fuller range of factors contributing to lowered intellect and school failure. In this larger perspective the health of the child and, in particular, his nutritional opportunities must assume a position of importance. It has long been recognized that the nutrition of the individual is perhaps the most ubiquitous factor affecting growth, health and development. Inadequate nutrition results in stunting, reduced resistance to infectious disease, apathy and general behavioral unresponsiveness. In a fundamental sense it occupies a central position in the multitude of factors affecting the child's development and functional capacity. It is therefore entirely understandable that, in a period dedicated to the improvement of man and his capacities, renewed attention has been directed to the relation of nutrition to intelligence and learning ability.

As is almost always the case in new areas of inquiry, clarity of thought and concept has not kept pace with zeal. Confusion has resulted from extravagent claims as to the unique contribution of malnutrition to brain impairment and intellectual deficit. Further confusion has been contributed by those who have with equal zeal sought to minimize the importance of nutritional factors and to argue for the primacy of social, genetic, cultural, or familial variables in the production of deficit. Little that is useful emerges from such sterile controversy. It is a truism that malnutrition occurs most frequently in those segments of the population who are economically, socially and culturally disadvantaged. When lowered intellect is demonstrated in malnourished children coming from such groups it is not difficult to ignore a consideration of the possible contribution of nutritional and health factors by pointing to the possibility that the children affected are dull because they are the offspring of dull parents; or that the general impoverishment of their environments has resulted in experiential deprivations sufficient to account for reduced intellectual function. Such an argument implies that the children are malnourished

because their parents are dull and that their functional backwardness stems from the same cause as their malnutrition. On logical grounds one could of course argue the very opposite from the same bodies of data. However, to do so would not be to consider the issue seriously but to engage in a debater's trick. The serious task is to disentangle, from the complex mesh of negative influences which characterize the world of disadvantaged children, the particular and interactive contributions which different factors make to the development of depressed functional outcomes. A responsible analysis of the problem, therefore, seeks to define the particular role which may be played by nutritional factors in the development of malfunction, and the interaction of this influence with other circumstances affecting the child.

Before considering the ways in which available research permit us to achieve this objective it is of importance to clarify the term malnutrition. Characteristically, we in the United States tend to react to the word in terms of a crisis model. When we think of malnutrition our imaginations conjure up images of the Apocalypse. We have visions of famines in India, of victims of typhoons, and of young Biafrans starved by war. These images reflect only a highly visible tip of an iceberg. Intermittent and marginal incomes as well as a technology which is inadequate to support a population result less often in the symptoms characteristic of starvation than in subclinical malnutrition or what Brock (1961) has called "dietary subnutrition . . . defined as any impairment of functional efficiency of body systems which can be corrected by better feeding." Such subnutrition when present in populations is reflected in stunting, disproportions in growth, and a variety of anatomic, physiologic, and behavioral abnormalities (Birch and Gussow, 1970). Our principal concern in this country is with these chronic or intermittent aspects of nutritional inadequacy.

In less highly developed regions of the world, and indeed in the United States as well, chronic subnutrition is not infrequently accompanied by dramatic manifestations of acute, severe, and if untreated lethal malnutrition particularly in infants and young children. These illnesses variously reflected in the syndromes of

marasmus, kwashiorkor, and marasmic-kwashiorkor are conditions deriving from acute exacerbations of chronic subnutrition which in different degrees reflect caloric deficiency, inadequacy of protein in the diet, or a combination of both states of affairs. Studies of children who recover from such disorders provide significant information on the effects of profound nutritional inadequacy on behavioral development.

In addition to the already mentioned conditions, malnutrition classically has been manifested as a consequence of the inadequate ingestion of certain essential food substances. The diseases of vitamin lack such as scurvy, rickets, pellagra, and beri-beri as well as the iron deficiency anemias are representative of this class of disorders.

None of the foregoing should be confused with the term hunger which often has been indiscriminately used as a synonym for malnutrition. Hunger is a subjective state and should not be used as the equivalent of malnutrition which is an objective condition of physical and physiologic suboptimum. Clearly, malnourished children may be hungry, but equally, hungry children may be well-nourished.

With these introductory considerations in mind we can now approach a series of questions. We shall be concerned with two issues: First, what is the state of sound knowledge of the relation of malnutrition in its various forms to intellect and learning and what is the significance of the evidence for psychology and education? And second, what are the implications of the evidence for improved functioning?

THE EVIDENCE

A number of model systems have been used to explore the relationship of malnutrition to behavior. At the human level these have consisted of: a) comparative studies of well and poorly grown segments of children in populations at risk of malnutrition in infancy; b) retrospective follow-up studies of the antecedent nutritional experiences of well-functioning and poorly-functioning children in such populations; c) intervention studies in which children in the poor risk population were selectively supplemented or unsupplemented during infancy and a comparative evaluation made of functioning in the supplemented and unsupplemented groups; d) follow-up studies of clinical cases hospitalized for severe malnutrition in early childhood; and e) intergenerational studies seeking to relate the degree to which conditions for risk of malnutrition in the present generation of children derived from the malnutrition or subnutrition experienced by their mothers when these latter were themselves children. Studies of human populations have been supplemented by a variety of animal models. These animal studies have been a) direct comparative follow-up investigations of the effects of nutritional difficulties in early life on subsequent behavioral competence and b) the study of the cumulative effects of malnutrition when successive generations of animals have been exposed to conditions of nutritional stress. The available evidence will be considered in relation to these investigative models.

In two of our reports (Cravioto, DeLicardie and Birch, 1966; Birch, 1971,) we have reviewed many of the earlier studies which have sought to explore the association between malnutrition and the development of intellect and learning. Perhaps the most complete study of the relation of growth achievement to neurointegrative competence in children living in environments in which severe malnutrition and chronic subnutrition are endemic is our study of Guatamalan rural Indian children. The children studied lived in a village having a significant prevalence level of both severe acute malnutrition and prolonged subnutrition during infancy and the preschool years. At school age relatively well-nourished children were identified as the better grown and children with the highest

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antecedent risk of exposure to malnutrition identified as those with the lowest growth achievements for age. On the basis of this reasoning two groups of children were selected from all village children in the age range six to 11 years. These groups encompassed the tallest and shortest quartiles of height distributions at each age for the total population of village children. In order to avoid problems associated with the use of intelligence tests as measures of functioning in pre-industrial communities, levels of development in the tall and short groups were compared by means of evaluating intersensory integrative competence by a method developed by Birch and Lefford (1963). In this method of evaluation children are required to judge whether geometric forms presented in different sensory modalities are the same or different. Competence in making such judgments follows a clearly defined developmental course in normal children in the age range studied.

At all ages taller children exhibited higher levels of neurointegrative competence than did the shorter group. Overall, the shorter children were two years behind their taller agemates in the competence which they exhibited in processing information across sensory systems.

In order to control for the possibility that height differences were reflecting differences in antecedent nutritional status rather than familial differences in stature, the child's height was correlated with that of the parents. The resulting correlation was extremely low and insignificant. This stands in marked contrast to the finding in the same ethnic group living in more adequate nutritional circumstances. Under these latter conditions the height of children correlates significantly with that of their parents.

Secondly, it was possible that the shorter children, in the community at risk as well as in communities not at risk of malnutrition, were merely exhibiting generalized developmental lag both for stature and for neurointegrative maturation. However, no differences in neurointegrative competence attached to differences in stature in the children not exposed to endemic malnutrition.

Finally, it was possible that the shorter children came from home environments significantly lower in socioeconomic status, housing and parental education, and that both the malnutrition and the reduced neurointegrative competence stemmed independently from these environmental deficits. When differences in these factors were controlled for they did not erase the differences in intersensory integrative competence between children of different growth achievements for age in the community at nutritional risk.

Over the past several years replications of this study have been conducted in Mexico by Cravioto and DeLicardie (1968), and in India by Champakam et al.(1968). In addition, Cravioto, Espinosa and Birch (1967) have examined another aspect of neurointegrative competence, and auditory-visual integration, in Mexican children of school age. Once again, in children in communities at risk of malnutrition, differences in growth achievement at school age were reflected in differences in auditory-visual integration favoring the taller children. These latter findings are of particular importance because of the demonstrated association between such competence and the ability to acquire primary reading skill (Birch and Belmont, 1964, 1965; Kahn and Birch, 1968).

A major consideration in interpreting the findings of all these studies is the fact that antecedent malnutrition is being inferred from differences in height rather than by direct observation of dietary intakes during the growing years. However, a multitude of data from earlier studies beginning with those of Boas (1910) on growth differences in successive generations of children of Jewish immigrants, of Boyd-Orr (1936) on secular trends in the height of British children, of Greulich (1958) on the height of Japanese immigrants, of Mitchell (1962, 1964) on the relation of nutrition to stature, of Bouterline-Young (1962) on Italian children, as well as the recent study of heights of 12 year old Puerto Rican boys in New York City by Abramowicz (1969) all support the validity of such an inference.

It should be noted too that findings similar to those obtained in the Guatamalan and Mexican studies have been reported by Pek Hien Liang et al. (1967) from Indonesia, and Stoch and Smythe

(1963, 1968) from South Africa. In the Indonesian study 107 children between five and 12 years of age were studied. All were derived from lower socioeconomic groups. Forty-six of these children had been classified as malnourished during a previous investigation into nutritional status in the area carried out some years earlier. All children were tested on the Wechsler Intelligence Scale for Children (WISC) and Goodenough tests. The scores showed a clear advantage for the better grown and currently better nourished children. Moreover, the data indicated that the shortest children were markedly overrepresented in the group that had been found to be malnourished in the earlier survey, and the largest deficits in IQ were found to be associated with the poorest prior nutritional status.

Stoch and Smythe have carried out a semi-longitudinal study of two groups of South African Negro children, one judged in early childhood to be grossly underweight due to malnutrition, and the other considered adequately nourished. At school age, the malnourished children as a group had a mean IQ which was 22.6 points lower than that of the comparison group. Moreover, these relative differences were sustained through adolescence. Unfortunately, interpretation of the findings in this study is made difficult because the better nourished children came from better families and had a variety of nursery and school experiences unshared by the poorly grown children.

Comparative studies of differential cognitive achievement in better and less well-nourished groups in communities at high levels of subnutrition have been supplemented by a relatively large number of follow-up evaluations of children who had been hospitalized for serious nutritional illness (marasmus or kwashiorkor) in infancy. As will be recalled from our earlier remarks, marasmus is a disorder produced by an insufficient intake of proteins and calories and tends to be most common in the first year of life. Kwashiorkor -- a syndrome produced by inadequate protein intake accompanied by a relatively adequate caloric level or in its marasmic form associated with reduced calories as well -- is more common in the post-weanling between nine months and two years of age.

As early as 1960, Waterlow, Cravioto and Stephen (1960) reported that children who suffered from such severe nutritional illnesses exhibited delays in language acquisition. In Yugoslavia, Cabak and Najdanvic (1965) compared the IQ levels of children hospitalized for malnutrition at less than 12 months of age with that of healthy children of the same social stratum and reported a reduced IQ in the previously hospitalized group. Of perhaps greater interest was their report of a significant correlation between the severity of the child's illness on admission -- as estimated in his deficit of expected weight for age -- and depression of IQ in the school years. Indian workers (Champakam, et al., 1968) studied many variables in a group of 19 children who between 18 and 36 months of age had been hospitalized and treated for kwashiorkor. When compared at school age with a well-matched control group, significantly depressed IQ was found in the children previously severely malnourished.

In order to control more fully for differences in the child's genetic antecedents and microenvironment which may still exist even when more general controls for social class and general circumstances are used in the selection of a comparison group, we (Birch, et al., 1971; Hertzig, Birch, Tizard and Richardson, 1971) have compared children previously malnourished in infancy with their siblings as well as with children of similar social background. In the first of these studies intelligence at school age was compared in 37 previously malnourished Mexican children and their siblings. The malnourished children had all been hospitalized for kwashiorkor between the ages of six and 30 months. The siblings had never experienced a bout of severe malnutrition requiring hospitalization. Sibling controls were all within three years of age of the index cases. Full scale WISC IQ of the index cases was 13 points lower than that of the sibling controls. Verbal and performance differences were of similar magnitude and in the same direction. All differences were significant at less than the 0.01 per cent level of confidence. These findings are in agreement with those of the Yugoslav and Indian workers and the use of sibling controls removes a potential contaminant for interpretation.

In the second study (Hertzig, Birch, Tizard and Richardson, 1971), a large sample of 74 male Jamaican children, who had been hospitalized for severe malnutrition before they were two years of age, were compared with their brothers nearest in age, and with their classmates whose birthdate was closest to their own. All children were between six and 11 years of age at follow-up. On examination, neurologic status, intersensory competence, intellectual level, and a variety of language and perceptual and motor abilities were evaluated. Intellectual level was significantly lower in the index cases than in either the siblings or the classmate comparison groups. As was to be expected, the order of competence placed the classmate comparison group at the highest level, the index cases at the lowest, and the sibs at an intermediate level. The depressed level of the siblings in relation to classmates suggests one disadvantage in sibling studies. Clearly, the presence of a child hospitalized for severe malnutrition identifies a family in which all children are at a high level of risk for significant undernutrition on a chronic basis; the index child merely represents an instance of acute exacerbation of this chronic marginal state. Therefore,the index cases and sibs are similar in that they share a common chronic exposure to subnutrition and differ only in that the index cases have experienced a superimposed episode of acute nutritional illness as well. Thus, the use of sibling controls, in fact, does not compare malnourished with non-malnourished children. Rather, it determines whether siblings who differ in their degree of exposure to nutritional risk differ in intellectual outcomes and supports the view that graded degrees of malnutrition result in graded levels of intellectual sequelae.

Other follow-up studies of acutely malnourished children such as those of Cravioto and Robles (1965) in Mexico, Pollitt and Granoff (1967) in Peru, Botha-Antoun, Babayan and Hafrouche (1968) in Lebanon, and Chase and Martin (1970) in Denver, have all been shorter-term follow-ups of younger children. Cravioto and Robles (1965) studied the developmental course of returning competence in children hospitalized for malnutrition during the period of their treatment and recovery while in hospital. Their findings indicated that behavioral recovery was less complete in the youngest children (hospitalized before six months of age)

than in older children. They posed the possibility that this earliest period of infancy was the one most critical for insult to developing brain and thus to eventual intellectual outcome. However, the above-mentioned study of Jamaican children did not yield findings which supported this possibility. In that study approximately equal numbers of children having experienced an acute episode of malnutrition in each of the four semesters of the first two years of life were examined. Equivalent depression of IQ was found to characterize each of the groups when these were separated by age at hospitalization (Hertzig, Birch, Tizard and Richardson, 1971.)

In the Lebanese and Peruvian, short-term follow-up studies noted above, depression in intellectual level tended to be found in the index cases. In the American study (Chase and Martin, 1970) and in a Chilean study (Monckeberg, 1968) the findings have shown depression in intellectual function in the pre-school years in children hospitalized for malnutrition during the first year of life. The American investigators working in Colorado found that 20 children who had been hospitalized for malnutrition before the age of one year had a mean developmental quotient on the Yale Revised Developmental Examination which was 17 points lower than that achieved by a matched control group of children who had not been malnourished. All of these studies strongly suggest that malnutrition of severe degree in early life tends to depress the intellectual functioning at later ages.

In summary, the follow-up studies of children who have been exposed to hospitalization for a bout of severe acute malnutrition in infancy indicate an association of significant degree between such exposure and reduced intellectual level at school age. The studies, involving careful social class controls and sibship comparisons, suggest that it is not general environmental deprivation but rather factors which are uniquely related to the occurrence of severe malnutrition that are contributing to a depression in intellectual outcome. However, there is some indication that different degrees of recovery may be associated with different post-illness conditions. Thus, urban and rural differences in intellectual outcomes are

reported in the sibship comparison studies of Jamaican children referred to above.

The fact of such an association provides strongly suggestive but by no means definitive evidence that malnutrition directly affects intellectual competence. As Cravioto, DeLicardie and Birch (1966) have pointed out, at least three possibilities must be considered in the effort to define a causal linkage. The simplest hypothesis would be that malnutrition directly affects intellect by producing central nervous system damage. However, it may also contribute to intellectual inadequacies as a consequence of the child's loss in learning time when ill, of the influences of hospitalization, and of prolonged reduced responsiveness after recovery. Moreover, it is possible that particular exposures to malnutrition at particular ages may in fact interfere with development at critical points in the child's growth course and so provide either abnormalities in the sequential emergence of competence or a redirection of developmental course in undesired directions. Although certain of these possiblities (such as hospitalization and post-illness opportunites for recovery) can be explored in children, others for moral and ethical reasons cannot. Thus, it is impermissible to establish appropriate experimental models either for interfering with development at critical periods, or for inducing brain damage. The approach to these problems requires either detailed analyses of naturally occurring clinical models or the development of appropriate animal investigations.

Animal models of the effects of malnutrition on brain and behavior have been used to study the issue with a degree of control that is quite impossible in human investigation. A series of pioneering investigations (Dobbing, 1964, Davison and Dobbing, 1966;Widdowson, 1966) have demonstrated that both severe and modest degrees of nutritional deprivation experienced by the animal at a time when its nervous system was developing most rapidly results in reduced brain size and in deficient myelination. These deficits are not made up in later life even when the animal has been placed on an excellent diet subsequent to the period of nutritional deprivation.

More recent studies by Zamenhof, et al. (1968) and by Winick (1968) have demonstrated that nutritional deprivation is also accompanied by a reduction in brain cell number. This latter effect has been demonstrated too in the human brains of infants who have died of severe early malnutrition (Winick and Rosso, 1969).

Enzymatic maturation and development in brain is also affected, and Chase et al. (1967, 1970) have demonstrated defective enzyme organization in the brains of malnourished organisms.

In all of these studies the evidence indicates that the effects of malnutrition vary in accordance with the time in the organism's life at which the deprivation is experienced. In some organisms the effects are most severe if the nutritional insult occurs in the prenatal period whereas in others this phenomenon occurs during early postnatal life.

Some confusion in the interpretation of evidence has occurred because of the use of different species, since in different organisms the so-called critical periods occur at different points in the developmental course. Thus, in pigs brain growth and differentiation is occurring most rapidly in the period prior to birth whereas in the rat the most rapid growth occurs when the animal is a nursling. In human beings the period for rapid growth is relatively extended and extends from mid-gestation through the first six through nine months of postnatal life. In man, the brain is adding weight at the rate of one to two mg/minute at birth and goes from 25 per cent of its adult weight at birth to 70 per cent of its adult weight at one year of age. After this age, growth continues more slowly until final size is achieved. Differentiation as well as growth occurs rapidly during the critical periods, with myelination and cellular differentiation tending to parallel changes in size.

Since brain growth in different species is occurring at different points in the life course, it is apparent that deprivations that are experienced at the same chronologic ages and life stages will have different effects in different species. Thus deprivation during early postnatal life will have little or no effect upon brain size and

structure in an organism whose brain growth has largely been completed during gestation. Conversely, intrauterine malnutrition is likely to have only trivial effects on the growth of the brain in species in which the most rapid period for brain development has occurred postnatally. When these factors are taken into account the data leave no doubt that the coincidence of malnutrition with rapid brain growth results in decreased brain size and in altered brain composition.

It would be unfortunate if brain growth in terms of cell number were to be viewed as the only definer of rapid change and thus of critical periodicity. In the human infant neuronal cell number is probably fully defined before the end of intrauterine life. Thereafter, through the first nine months of postnatal life, cell replication is that of glial cells, a process which terminates by the end of the first year. However, myelination continues for many years thereafter as does the proliferation of dendrite branchings and other features of brain organization. It is most probable, therefore, that in man the period of vulnerability extends well beyond the first year of life and into the pre-school period. Such a position is supported by the findings of Champakam et al. (1968). These workers, it will be recalled, found significant effects on intellect in their group of malnourished children who had experienced severe malnutrition when they were between 18 and 36 months of age.

Other workers who have used animal models have sought to study the effects of malnutrition on behavioral outcomes rather than on brain structure and biochemical organization. The typical design of these studies are investigations in which animals have been raised upon diets which were inadequate with respect to certain food substances, or, in which general caloric intake has been reduced without an alteration in the quality of the nutriments. Such animals have then been compared with normally nourished members of the species with respect to maze learning, avoidance conditioning, and open field behavior. Unfortunately, most of the investigations have suffered from one or another defects in design which make it difficult to interpret the findings. Though in general the nutritionally deprived organisms have tended to be disadvantaged as learners, it is not at all clear whether this is the result of their food lacks at

critical points in development or whether the differences observed stem from the different handling, caging, and litter experiences to which the well and poorly nourished animals were exposed. Moreover, in a considerable number of studies food or avoidance motivation have been used as the reinforcers of learning. There is abundant evidence (Mandler, 1958; Elliott and King, 1960; Barnes, et al., 1968; Levitsky and Barnes, 1969) that nutritional deficiency in early life affects later feeding behavior. Consequently, it is difficult to know whether the early deprivation has affected food motivation or whether it has affected learning capacity. The use of learning situations which do not involve food but are based upon aversive reinforcement do not remove difficulties for interpretation since early malnutrition modifies sensitivity to such negative stimuli (Levitsky and Barnes, 1970).

Therefore, at present, one must recognize that although the animal evidence suggests that early malnutrition may influence later learning and behavior, it is by no means conclusive. Moreover, when learning has been deleteriously affected, the mechanism through which this effect has been mediated is by no means clear. What is required is a systematic series of experiments in which behavioral effects are more clearly defined, and in which the use of proper experimental designs, accompanied by appropriate controls, permits the nature of the mechanisms affected to be better delineated.

Thus far, in our consideration of both the human and animal evidence, we have been considering the direct affects of nutritional deprivation on the developing organism. Clearly, this is too lir ted a consideration of the problem. It has long been known (Boyd-Orr, 1936) that nutritional influences may be intergenerational and that the growth and functional capacity of an individual may be affected by the growth experiences and nutrition of his mother. In particular, the nutritional history of the mother and its effect upon her growth may significantly affect her competence as a reproducer. In turn, this reproductive inadequacy may affect the intrauterine and birth experiences of the offspring.

Bernard (1952) has clearly demonstrated the association between a woman's nutritional history and her pelvic type. He compared one group of stunted women in Aberdeen, Scotland with well-grown women and found that 34 per cent of the shorter women had abnormal pelvic shapes conducive to disordered pregnancy and delivery as compared with seven per cent of the well-grown women with whom they were compared. Gruelich, Thoms and Twaddle (1939) still earlier had reported that the rounded or long oval pelvis, which appears to be functionally superior for childbearing, was more common in well-off, well-grown women than in economically less privileged clinic patients. They further noted, as had Bernard, that these pelvic abnormalities were strongly associated with shortness.

Sir Dugald Baird and his colleagues in the City of Aberdeen, Scotland, have conducted a continuing series of studies from 1947 onward on the total population of births in this city of 200,000 in an effort to define the patterns of biologic and social interactions which contribute to a woman's growth attainments and to her functional competence in child-bearing. More than 20 years ago Baird (1947) noted that short stature, which was five times as common among lower class women than among upper class women, was associated with reproductive complications. He pointed out (1949), on the basis of analyzing the reproductive performances of more than 13,000 first deliveries, that fetal mortality rates were more than twice as high in women who were under five feet one inch in height than in women whose height was five feet four inches or more. Baird and Illsley (1953) demonstrated that premature births were almost twice as common in the shorter than in the taller group. Thomson (1959) extended these observations by analyzing the relation between maternal physique and reproductive complications for the more than 26,000 births which had occurred in Aberdeen over a ten year period and found that short stature in the mother was strongly associated with high rates of prematurity, delivery complications and perinatal deaths at each parity and age level. He concluded that "it is evident that whatever the nature of the delivery the fetus of a short woman has less vitality and is less likely to be well-grown and to survive than that of a tall woman."

It was of course possible that these findings simply reflected differences in social class composition of short and tall women and were based upon differences in "genetic pool" rather than in stunting as such. To test this hypothesis the Aberdeen workers (Baird, 1964) re-examined their data for perinatal mortality and prematurity rates by height within each of the social classes for all Aberdeen births occurring in the ten year period from 1948 to 1957. They found that shortness in every social class was associated with elevated rates of both prematurity and perinatal deaths. Concerned that the findings in Aberdeen might not be representative they also analyzed the data from the all-Britain perinatal mortality survey of 1958 and confirmed their findings. Moreover, Thomson and Billewicz (1963) in Hong Kong and Baird (1964) have substantiated the Aberdeen findings for Chinese and West African women, respectively. Other findings in a similar vein from this series have been summarized by Illsley (1967).

The available data therefore suggest that women who are not well-grown have characteristics which negatively affect them as childbearers. In particular, short stature is associated with pregnancy and delivery complications and with prematurity. Since growth achievement within ethnic groups is a function of health history and in particular nutrition, it is clear that the mother's antecedent nutritional history when she herself was a child can and does significantly influence the intrauterine growth, development and vitality of her child. Moreover, an inadequate nutritional background in the mother places this child at elevated risk for damage at delivery.

It is instructive to consider the consequences for mental development and learning failure that attach to the most frequently occurring consequence of poor maternal growth -- prematurity. Concern with the consequences of this condition is hardly new. Shakespeare indicted it as one element in the pecularities of Richard III, and Little (1862) linked it with the disorder we now call cerebral palsy. Benton (1940) reviewed the literature up to 1940 and found that, though most students of the problem maintained that prematurity was a risk to later mental development, others could find no negative consequence attaching to it. At that

time no resolution of disagreement could be made because most of the early studies had been carried out with serious deficiencies in design and in techniques of behavioral evaluation. Groups who were of low birth weight or early in gestational age were often compared with full-term infants who differed from them in social circumstances as well as in perinatal status. Estimates of intellectual level were made with poor instruments and often dependent on "clinical impression" or testimony from parents or teachers.

Serious and detailed consideration of the consequences of low birth weight for later behavioral consequences can properly be said to have been begun by Pasamanick, Knobloch and their colleagues shortly after World War II. These workers were guided by a concept which they referred to as a "continuum of reproductive casualty." They argued that there was a set of pregnancy and delivery complications which resulted in death by damaging the brain and hypothesized that in infants who survived exposure to these risks "there must remain a fraction so injured who do not die, but depending on the degree and location of trauma, go on to develop a series of disorders extending from cerebral palsy, epilepsy and mental deficiency, through all types of behavioral and learning disabilities, resulting from lesser degrees of damage sufficient to disorganize behavioral development and lower thresholds to stress" (Pasamanick and Knobloch, 1960). In a series of retrospective studies these investigators identified prematurity and low birth weight as being among the conditions most frequently associated with defective behavioral outcomes. They therefore, in association with Rider and Harper, undertook a prospective study of a balanced sample of 500 premature infants born in Baltimore in 1952 and compared them with full-term control infants born in the same hospitals who were matched with the prematures for race, maternal age, parity, season of birth and socioeconomic status (Knobloch, et al., 1956.) Four hundred pairs of cases and controls were still available for study when the children were between six and seven years of age, and examination of the sample indicated that at this age the prematures and full term children continued to be matched for maternal and social attributes (Wiener, et al., 1965). Findings at various ages persistently showed the prematures to be less intellectually

competent than the controls. At ages three to five the prematures were relatively retarded intellectually and physically and had a higher frequency of definable neurological abnormalities (Knobloch, et al., 1959; Harper, et al., 1959). At ages six through seven IQ scores on the Stanford-Binet test were obtained as were WISC IQ's at ages eight to nine. At both age levels lower birth weights were associated with lower IQ's (Wiener, et al., 1965, 1968).

Although certain British studies such as that of McDonald (1964) and of Douglas (1956, 1960) appear to be somewhat discrepant with these findings, reanalysis of their data (Birch and Gussow, 1970) indicates a similar trend. More dramatic differences between prematures and full term infants have been reported by Drillien (1964, 1965) but interpretation of her data is made difficult by complexities in the selection of the sample studied.

A number of analyses suggest that the effects of prematurity are not the same in different social classes. Children from the lowest social classes appear to have subsequent IQ and school performances more significantly depressed by low birth weight than is the case for infants in superior social circumstances. This has been reported for Aberdeen births (Illsley, 1966; Richardson, 1968) and for Hawaiian children in the Kauai pregnancy study of Werner (1967). There appears to be an interaction between birthweight and family social condition in affecting intellectual outcome, but the precise mechanisms involved in this interaction are as yet unclear.

If the risk of deficient intellectual outcome in prematurity is greatest for those children who are otherwise socially disadvantaged as well, our concern in the United States with the phenomenon of prematurity must be increased. In 1962, more than 19 per cent of non-white babies born in New York City had a gestational age of less than 36 weeks as compared with 9.5 per cent of white babies, and in Baltimore this comparison was 25.3 per cent in non-white infants as compared with 10.3 per cent in whites (National Center for Health Statistics, 1964). In 1967,

nationally, 13.6 per cent of non-white infants weighed less than 2,500 grams as compared with 7.1 per cent of white infants (National Center for Health Statistics, 1967). Other relevant and more detailed analyses of the social distribution of low birth weight and gestational age on both national and regional bases, together with an analysis of their secular trends, provides additional support for these relationships (Birch and Gussow, 1970). Thus prematurity is most frequent in the very groups in which its depressing affects on intelligence are greatest.

On the basis of the evidence so far set forth, it may be argued with considerable justification that one can reasonably construct a chain of consequences starting from the malnutrition of the mother when she was a child, to her stunting, to her reduced efficiency as a reproducer, to interuterine and perinatal risk to the child, and to his subsequent reduction in functional adaptive capacity. Animal models have been constructed to test the hypotheses implied in this chain of associations, most particularly by Chow and his colleagues (Chow, et al., 1968; Hsueh, et al., 1967), as well as by Cowley and Griesel (1963, 1966). The findings from these studies indicate that second and later generation animals who derive from mothers who were nutritionally disadvantaged when young, are themselves less well-grown and behaviorally less competent than animals of the same strain deriving from normal mothers. Moreover, the condition of the offspring is worsened if nutritional insult in its own life is superimposed on early maternal malnutrition.

A variety of factors would lead us to focus upon the last month of intrauterine life as one of the "critical" periods for the growth and development of the central nervous system in humans. Both brain and body growth together with differentiation are occurring at a particularly rapid rate at this time. It has been argued, therefore, that whereas marginal maternal nutritional resources may be sufficiently adequate to sustain life and growth during the earlier periods of pregnancy, the needs of the rapidly growing infant in the last trimester of intrauterine existence may outstrip maternal supplies. The work of Gruenwald et al.

(1963), among others, suggests that maternal conditions during this period of the infant's development are probably the ones which contribute most influentially to low birth weight and prematurity. Such concerns have led to inquiries into the relation of the mother's nutritional status in pregnancy to the growth and development of her child. In considering this question, it is well to recognize that, as yet, we have no definitive answer to the question of the degree to which maternal nutrition during pregnancy contributes to pregnancy outcome. Clearly, whether or not nutritional lacks experienced by the mother during pregnancy will affect fetal growth is dependent upon the size and physical resources of the mother herself. Well-grown women are most likely to have tissue reserves which can be diverted to meet the nutritional needs of the fetus even when pregnancy is accompanied by significant degrees of contemporary undernutrition. Conversely, under the same set of circumstances, poorly grown women with minimal tissue reserves could not be expected to be able to provide adequately for the growing infant.

Children coming from families in which the risks for exposure to malnutrition are high are unlikely to experience nutritional inadequacies only in early life. It is far more likely that earlier nutritional inadequacies are projected into the preschool and school years. Such a view receives support from numerous surveys as well as from recent testimony presented before the Senate Committee on Nutrition and Human Needs (1968-70). Our knowledge of the degree to which children and families at risk continue to be exposed to nutritional inadequacies derives from a series of indirect and direct methods of inquiry. At an indirect level it can be argued that family diet in the main is very much dependent upon family income level. The report Dietary Levels of Household in the United States (1968) published by the United States Department of Agriculture underscores this proposition. According to a household survey conducted in the spring of 1965, only nine per cent of families with incomes of $10,000 and over a year were judged as having "poor" diets. However, the proportion of poor diets increased regularly with each reduction in income level. Eighteen per cent of the families in the income range $5,000 to $6,999 had poor diets

whereas 36 per cent of families earning under $3,000 a year reported poor diets, that is, diets containing less than two-thirds of the recommended allowance of one or more essential nutrients. Conversely, the proportion of "good" diets went from 63 per cent in the $10,000-and-over category down to 37 per cent in the under $3,000 group. Of course, income alone is not an adequate indicator of socioeconomic status since in families with equal incomes more education appears to produce a better diet (Jeans, Smith and Stearns, 1952; Murphy and Wertz, 1954; Hendel, Burke, and Lund, 1965). But, at the least such figures suggest that we must be seriously concerned with just how badly nourished are our poor in what we often claim is the "best-fed nation in the world."

Reports of the survey type may be supplemented by inquiries in which mothers are asked what they feed their families and how much of what kinds of food they purchase. Similarly actual food intakes may be estimated by requests for the retrospective recall of all foods eaten over the last 24 hours. Owen and Kram (1969), in a study of nutritional status in Mississippi preschool children, found not only that the poorer children were on the average smaller than more affluent children but that their diets were significantly low in calories, vitamin C, calcium and riboflavin. Dibble, et al. (1968) in Onondaga County, New York found that, among students from a junior high school which was 94 per cent Negro and predominantly laboring class, 41 per cent had come to school without breakfast; however in two "overwhelmingly white" junior high schools, only seven per cent in one school and four per cent in the other had skipped breakfast. In recent studies among teen-agers in Berkeley, California, Hampton et al., (1967) and Huenemann et al., (1968) found that intakes of all nutrients declined with socioeconomic status, and that Negro girls and boys had worse intakes than those in other ethnic groups. Huenemann also found that, among junior and senior high school students studied over a two year period, 90 per cent of the Negro teen-agers had irregular eating habits and many appeared to be "fending for themselves."

Christakis et al. (1968) who have carried out the first dietary study of New York school children in 20 years found that, in an economically depressed district, the diets of 71 per cent of the children examined were poor and that less than 7 per cent had excellent diets. Moreover, his data demonstrated that if the child's family were on welfare the likelihood of his having a poor diet was much increased.

The situation is not markedly different in the Roxbury district of Boston. In this area Myers et al. (1968) studied the diets and nutritional status of 4th, 5th and 6th graders about two thirds of whom were black. Meals were ranked as "satisfactory" or "unsatisfactory." Four satisfactory ratings for a given meal over the four day period produced a "satisfactory" rating for the meal. Fifty-five per cent of the children failed to get such a satisfactory rating for breakfast, 60 per cent of them did not have satisfactory lunches, and 42 per cent had less than four satisfactory evening meals in four days. "Satisfactory" scores declined with age for all meals, and Negroes generally had more unsatisfactory ratings than Caucasians. The schools had no school-lunch programs, and lunches were the poorest meals. One-third of the children had two or more unsatisfactory lunch ratings in four days. During the four day period, 64 per cent of the children had less than two glasses of milk a day, 132 children had no citrus fruit, and only one child had a green or yellow vegetable; 37 per cent of the Negro and 46 per cent of the Caucasian children had "unsatisfactory" intakes of the protein foods in the meat, fish, poultry, eggs, and legume group. "It is evident," the authors concluded," that many of these children were eating poorly."

These data are illustrative and not atypical of the national picture. The preliminary reports deriving from the National Nutrition Survey serve to confirm these findings on a national scale. The evidence, though scattered and of uneven quality, strongly indicates that economically and ethnically disadvantaged children eat poorly in both the pre-school

and school age periods.

Direct clinical studies, occurring largely within the Head Start Program, serve to support the impression produced by the data of nutritional surveys. One way of examining possible sub-nutrition on an economical clinical basis is to define the prevalence of iron-deficiency anemia. Hutcheson and Wright (1968), reporting on a very large sample of poor white and Negro children in rural Tennessee, found the highest level of anemia among children around one year old. Of the whole group of 15,681 children up to six years of age, 20.9 per cent had hematocrits of 31 per cent, indicating a marginal status. Among the year-old children, however, the incidence of low hematocrits was even higher; 27.4 per cent of the whites and 40 per cent of the nonwhites had hematocrits of 31 per cent or less, and ten per cent of the whites and one-quarter of the nonwhites had hematocrits of 30 per cent or under, indicating a more serious degree of anemia. Low hemoglobin level was also most common among the younger children in a group Gutelius (1969) examined at a child health center in Washington, D.C. Iron-deficiency anemia, determined by hemoglobin level and corroborative red cell pathology, was found among 28.9 per cent of the whole group of 460 Negro preschoolers, but children in the age group 12-17 months had a rate of anemia of 65 per cent. Gutelius points out, moreover, that these were probably not the highest-risk children, since the poorest and most disorganized families did not come for well-baby care at all, and of those who did attend, the test group included only children who had not previously had a hemoglobin determination -- that is, they were children judged to be "normal" by the clinic staff. Thus "many of the highest-risk children had already been tested and were not included in this series."

Even in the summer 1966 Head Start program, in which the incidence of other disorders was surprisingly low (North, 1967), studies indicated that 20-40 per cent of the children were

suffering from anemia, a proportion consistent with the findings of various studies summarized by Filer (1969) as well as with the level of anemia found in a random sample of predominantly lower class children coming into the pediatric emergency room of the Los Angeles County Hospital (Wingert, 1968). Anemia rates as high as 80 per cent among pre-school children have been reported from Alabama (Mermann, 1966) and Mississippi (Child Development Group of Mississippi, 1967).

It is clear from such evidence that some degree of malnutrition is relatively widespread among poor children; however, we have already seen that the effects of inadequate nutrition on growth and mental development depend to a very large extent on the severity, the timing, and the duration of the nutritional deprivation. Inadequate as are our data on the true prevalence of malnutrition among children in this country, we are even less informed about its onset or about its severity and quality. The absence of such knowledge must not be taken to reflect the absence of the problem but rather the lack of attention which has been devoted to it.

IMPLICATIONS, PROGRAMS AND PROBLEMS

The evidence we have surveyed strongly indicates that nutritional factors at a number of different levels contribute significantly to depressed intellectual level and learning failure. These effects may be produced directly as the consequences of irreparable alterations of the nervous system or indirectly as a result of ways in which the learning experiences of the developing organism may be significantly interfered with at critical points in the developmental course.

If one were to argue that a primary requirement for normal intellectual development and for formal learning is the ability to process sensory information and to integrate such information across sense systems the evidence indicates that both severe acute malnutrition in infancy as well as chronic subnutrition from birth into the school years results in defective information processing. Thus, by inhibiting the development of a primary process essential for certain aspects of cognitive growth, malnutrition may interfere with the orderly development of experience and contribute to a suboptimal level of intellectual functioning.

Moreover, an adequate state of nutrition is essential for good attention and for appropriate and sensitive responsiveness to the environment. One of the most obvious clinical manifestations of serious malnutrition in infancy is a dramatic combination of apathy and irritability. The infant is grossly unresponsive to his surroundings and obviously unable to profit from the objective opportunities for experience present in his surroundings. This unresponsiveness characterizes his relation to people as well as to objects. Behavioral regression is profound, and the organization of his functions are markedly infantalized. As Dean (1960) has put it, one of the first signs of recovery from the illness is an improvement in mood and in responsiveness to people -- "the child who smiles is on the road to recovery."

In children who are subnourished one also notes a reduction in responsiveness and attentiveness. In addition, the subnourished child is easily fatigued and unable to sustain either prolonged physical or mental effort. Improvement in nutritional status is accompanied by improvements in these behaviors as well as in physical state.

It should not be forgotten that nutritional inadequacy may influence the child's learning opportunities by yet another route, namely, illness. As we have demonstrated elsewhere (Birch and Cravioto, 1968; and Birch and Gussow, 1970), nutritional inadequacy increases the risk of infection, interferes with immune mechanisms, and results in illness which is both more generalized and more severe. The combination of subnutrition and illness reduces time available for instruction and so, by interfering with the opportunities for gaining experience, disrupts the orderly acquisition of knowledge and the course of intellectual growth.

We have pointed also to intergenerational affects of nutrition upon mental development. The association between the mother's growth achievements and the risk to her infant is very strong. Poor nutrition and poor health in the mother when she was a girl result in a woman at maturity who has a significantly elevated level of reproductive risk. Her pregnacy is more frequently disturbed and her child more often of low birth weight. Such a child is at increased risk of neurointegrative abnormality and of deficient IQ and school achievement.

Despite the strength of the argument that we have developed, it would be tragic if one were now to seek to replace all of the other variables -- social, cultural, educational, and psychological -- which exert an influence on intellectual growth with nutrition. Malnutrition never occurs alone. It occurs in conjunction with low income, poor housing, familial disorganization, a climate of apathy, ignorance and despair. The simple act of improving the nutritional status of children and their families will not and cannot of itself fully solve the problem of intellectual deficit and school failure. No single improvement in conditions will have this result. What must be recognized rather is that within our overall effort to improve the

condition of disadvantaged children nutritional considerations must occupy a prominent place and, together with improvements in all other facets of life -- including relevant and directed education -- contribute to the improved intellectual growth and school achievement of disadvantaged children.

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